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## Research report

# ‘Side effects’ of ECT are mainly depressive phenomena and are independent of age

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## Abstract

**Background:** The aetiology of reported side effects of electroconvulsive therapy (ECT) is unclear. We examined the interaction of depression and age on adverse neuropsychological and putative side effects of ECT. **Method:** Inpatients ( $N = 81$ ; median age 70 years) with major depression were assessed prospectively pre-ECT, immediately post-ECT and 1–3 years later. Patients were administered the Hamilton Rating Scale for Depression (HRSD), the Global Assessment of Functioning scale (GAF) and neuropsychological tests from the Wechsler Memory Scale. Side effects and total burden scores were rated pre- and post-treatment. **Results:** HRSD and GAF scores improved with treatment after ECT, but the prevalence and total burden of side effects were unchanged. Side effect burden was related to depression level before and after ECT. Improvement in depression correlated with reduction in side effect burden. There was a significant decline in side effect burden after controlling for change in depression. Patients’ scores on neuropsychological measures did not appear to change after ECT or between pre-ECT and follow-up. Re-analysis, allowing for age, chronicity of depression, medication use and development of dementia, did not alter the findings. **Limitations:** lack of a control group, lack of information on ECT technique, incomplete data sets and limited neuropsychological testing. **Conclusions:** ECT, an effective treatment for depression, does not cause significant side effects or neuropsychological impairment, which are more likely to be depressive phenomena. ECT appears to be safe for old ( $\geq 65$  years) and very old ( $\geq 75$  years) patients, who do not appear to be more susceptible to adverse effects. © 2001 Elsevier Science B.V. All rights reserved.

**Keywords:** Electroconvulsive therapy; Side effects; Cognition; Age; Aging; Depression

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## 1. Introduction

Fears of brain damage, cognitive impairment and physical side effects feature prominently in public perceptions of electroconvulsive therapy (ECT) (Fink, 1991; Coffey and Weiner, 1990; Baxter et al.,

1986). However, many of these concerns may be excessive and there are few empirical data to support such public concerns. In a recent review, Calev et al. (1995) concluded that standard courses of ECT leave little or no enduring cognitive side effects. Aside from retrograde amnesia concerning events in the week or two preceding treatment, there appear to be little or no enduring effects on memory (Benbow, 1989). Other cognitive faculties appear to be unaffected (Pisvejc et al., 1998; Calev et al., 1995) or even improved in the week following ECT, for example attention, reaction time and immediate learning (Weeks et al., 1980; Sobin et al., 1995).

ECT may result in side effects such as headache, nausea, tiredness and muscle aches and pains (e.g., Weiner et al., 1994; Fraser and Glass, 1980). We propose, as did Devanand et al. (1995), that many of these complaints are common before ECT and may be associated with the depressive disorder itself or factors associated with the depression such as the cerebrovascular disease purported to underpin depression beginning in late-life (Hickie et al., 1995; Hickie and Scott, 1998). Few studies have accounted for the possibility of persistent depressive symptoms in their reporting of side effects, and this considerably limits interpretation of the results. Much of the previous research in this area has been limited by retrospective methodologies and by poor consideration of pre-treatment data. For example, returning to a pre-ECT level of functioning does not mean that the effects of ECT have recovered, it only means that the effects of ECT are not larger than any overlapping symptoms of the initial depression (Calev et al., 1995).

Age has been consistently related to greater severity of post-ictal disorientation, confusion and cardiovascular complications (Cattan et al., 1990; Benbow, 1989; Alexopoulos et al., 1984), and may also be associated with greater memory dysfunction following treatment (Zervas et al., 1993; Benbow, 1989). The influence of age on other side effects is less apparent, although it appears that ECT is relatively safe in older patients (Weiner, 1982) and is associated with improved global functioning independent of age (Tew et al., 1999). It appears that the elderly may be more susceptible to adverse cognitive but not other side effects. Studies of cognitive function have reported a slight decrease in MMSE in

patients tested within 72 hours of completing their course of ECT (Tew et al., 1999) or an improvement if tested 1 week after completing ECT (Wilkinson et al., 1993). In both studies there was no differential effect with age, nor is there evidence that adverse cognitive effects endure, should they occur.

In a prospective, longitudinal study investigating the effects of ECT, patients were assessed before ECT, directly after treatment and 1 to 3 years later. We aimed to determine what were the effects of ECT and depression on neuropsychological and other purported adverse effects of ECT. We also investigated whether there was an interaction with age and the occurrence of side effects.

Our hypotheses were that purported adverse effects would (a) be fewer and less severe directly after ECT and on longer term follow-up than before ECT; (b) be related to measures of depression; and (c) be independent of age.

## 2. Method

### 2.1. Design and subjects

The methods used in the present study have been described elsewhere (Brodaty et al., 2000). One hundred and one consecutive inpatients who were to have ECT were recruited over a 2 year period from eight Sydney hospitals. Thirteen patients with primary organic disorders and seven with atypical/schizophrenic psychoses were excluded, leaving 81 patients with a primary diagnosis of major depressive episode as defined by DSM-III-R. Dementia was an exclusionary criterion. Each diagnosis was made by the treating psychiatrist and was confirmed independently by two other psychiatrists experienced in the assessment of mood disorders (IH, CM). Patients provided consent after being interviewed by a member of the research team, informed about the purpose of the study and given written information about the study.

### 2.2. Assessment

Subjects were initially assessed up to 1 week before commencing a course of ECT. A detailed medical and psychiatric history and information

about current psychiatric treatment were obtained from all patients. Diagnoses were made using a structured interview from which DSM-III-R diagnoses of depression and melancholic and psychotic sub-types could be generated (Brodaty et al., 1987). Duration of episode since onset, number of previous episodes of depression, and current medications were recorded. Depression severity was quantified by the structured 21-item Hamilton Rating Scale for Depression (HRSD; the higher the score, the greater the severity, Williams, 1988). Prior to the commencement of treatment, the interviewer inquired about 20 adverse events based on reports in the literature as occurring after ECT (Sackeim et al., 1987). These were each scored as: 0, not present; 1, mild; 2, moderate; or 3, severe. We report the total number of items affirmed and the total adverse event burden for each patient which is the sum of the scores for each adverse event.

A number of neuropsychological tests were administered pre-ECT. The Wechsler Memory Scale Associate Learning sub-test (AL), a measure of verbal learning (Wechsler, 1945), was scored as the sum of credits on easy associations divided by two plus the credits on hard associations. The Symbol-Digit Modalities Test (SDMT), a measure of psychomotor speed, attention and concentration (Smith, 1982), was scored as the total number of correct symbol substitutions during a 90 second period. Simple and complex reaction times, which measure psychomotor and decision speed (Huppert et al., 1987), and the time taken to complete the Trail Making Test (part A), a test of speed for visual search, attention and motor function (Reitan, 1958), were recorded in seconds. The Controlled Oral Word Association Test (COWAT), a measure of word fluency (Spreen and Benton, 1969), was scored as the total number of allowable words reported for the three stimulus letters. Age adjusted scores are presented.

Neuropsychological data pre-ECT were complete for 22 (27%) patients, partially complete for 44 (54%) patients and absent for 15 (19%) patients because they either refused or were too severely impaired to undertake testing. Post-ECT, 42 patients (51.9%) had complete, 16 (19.7%) partially complete, and 23 (28.4%) no neuropsychological data; while at follow-up 27 (33.3%) had complete, 18

(22.2%) partially complete and 36 (44.4%) no neuropsychological data. The sub-samples of the total ECT population ( $n = 81$ ) who had had neuropsychological testing were representative. Subjects who did and did not have neuropsychological testing prior to ECT ( $n = 66$  tested and 15 not tested), after ECT ( $n = 53$  and 13, respectively) and at follow-up ( $n = 33$  and 33, respectively) were similar as regards age, sex, index Hamilton depression score, duration of depression prior to ECT and number of ECT treatments.

Eighty subjects were reassessed by an independent research psychiatrist within 2 weeks following their final ECT, by use of the HRSD and the GAF. The adverse event questions and neuropsychological assessment were repeated post-ECT (except for an 81-year-old lady with psychotic depression who died after one ECT. Her death from septicaemia probably resulted from an infected site of intravenous cannulation which was inserted prior to ECT when she developed paralytic ileus as a complication of her depression, inanition and medication). Otherwise, post-ECT outcome data were complete.

At least 12 months (range 57 to 176 weeks, mean 109.9, S.D. 31.5) after the index ECT, subjects were contacted for follow-up interviews by another psychiatrist, not involved in earlier ratings. Depression severity and neuropsychological function (but not the side effect inventory) were reassessed. Those who refused further participation were asked for permission to obtain information from relevant health professionals and/or family members.

At follow-up, seven subjects had died, including two by suicide, one subject refused follow-up assessment and two could not be traced. Follow-up clinical outcomes were rated directly ( $n = 64$ ) or from collateral sources ( $n = 7$ ) for the remaining 71 subjects; GAF scores were rated for 73 subjects (including two who had suicided and were assigned a GAF score of 0); and HRSD scores were rated for 59 subjects. Twelve subjects were not assigned HRSD scores because their responses on that scale were judged to be unreliable ( $n = 3$ ), they only allowed a brief phone interview ( $n = 2$ ), information was only available from a relative or caregiver ( $n = 4$ ), or because they died more than 12 months after ECT but before the follow-up interview could be conducted ( $n = 3$ ). In the last case, GAF and clinical

outcome ratings were made on the basis of collateral information (e.g., nursing home staff, family reports, GP reports, medical file review) and represented subjects' level of functioning in the weeks prior to their terminal illness.

Subjects were recorded as demented at follow-up if they (i) met DSM-III-R criteria for dementia, (ii) obtained a Mini-Mental State (Folstein et al., 1975) score of less than 24 in the absence of delirium or concurrent depression, and/or (iii) had clear evidence of progressive cognitive and functional decline after the index episode. In addition, we required that this diagnosis be confirmed at further follow-up according to DSM-IV criteria and ratings of at least moderately severe dementia on the Clinical Dementia Rating (CDR  $\geq 2$ ) scale (Hughes et al., 1982). This second confirmatory follow-up occurred a mean of 64.3 months (S.D. 5.8, range 52.0–70.0) after the first follow-up.

### 2.3. Statistical analyses

A paired samples *t*-test was used to compare pre- and post-treatment GAF scores and a Wilcoxon matched pairs test was used to compare HRSD scores, which were significantly skewed, between pre and post. A Fisher's exact test was used for a two-by-two comparison between high and low HRSD scorers between pre- and post-ECT, since expected frequencies were lower than five. We calculated the effect sizes for change in depression and side effect burden by subtracting the mean scores post-ECT from the mean scores pre-ECT and dividing by the pooled standard deviation (Howell, 1987). Positive effect sizes represented improvement.

The side effects data from pre- to post-ECT were analysed using chi-square tests. Bonferroni adjustments were made to control for the number of paired comparisons (0.05/20). Point-biserial correlations were used to analyse demographic data involving dichotomous variables. Spearman correlations were used for comparisons involving Hamilton scores that were significantly positively skewed. A repeated measures ANOVA was used to compare pre- and post-ECT side effect burden when change in depression was controlled for.

Repeated measures ANOVAs were conducted on

each of the neuropsychological variables to compare pre-ECT with post-ECT scores and post-ECT with follow-up scores. Analyses were repeated after exclusion of patients who subsequently developed dementia. Bonferroni adjustments were made to control for the number of paired comparisons (0.05/10).

## 3. Results

### 3.1. Demographic and clinical details

The sample consisted of 28 men and 53 women, whose mean age was 67.2 years (S.D. 13.1, range 25–89, aged  $\geq 70 = 50.6\%$ ) and had had an average of 9.5 (S.D. 2.4) years of education. There were 81 patients assessed post-treatment and 71 at follow-up. Patients received a mean of 11.3 (S.D. 5.1) treatments. Sixty-eight percent of treatments were unilateral, seven percent bilateral and 25 percent mixed unilateral and bilateral.

### 3.2. Improvement in depression

Hamilton scores improved substantially with treatment (pre-ECT mean 28.8, S.D. 8.3; post-ECT mean 10.3, S.D. 9.0; Wilcoxon  $z = -7.7$ ,  $P < 0.001$ ). There were 75 (92.6%) patients with a HRSD score  $\geq 16$  before, and 22 (27.5%) patients with a HRSD score  $\geq 16$  following treatment ( $\chi^2 = 2.3$ ,  $P = 0.33$ ). Global assessment of functioning scores also improved from pre- to post-ECT (pre-ECT: mean 36.2, S.D. 15.1; post-ECT: mean 64.9, S.D. 16.5;  $t = -11.3$ ,  $P < 0.001$ ).

There was a significant decrease between pre- and post-treatment in the number of patients receiving antipsychotic medication ( $\chi^2 = 5.68$ , d.f. 1,  $P = 0.02$ ), although not in the number of patients receiving tricyclic or tetracyclic medication ( $\chi^2 = 0.46$ , d.f. 1,  $P = 0.5$ ) (Table 1). We now report side effects and neuropsychological outcome separately.

### 3.3. Side effects

Our first hypothesis was supported: the number and levels of side effects did not increase after ECT (Table 2). There was a small, insignificant reduction

Table 1  
Number and proportion of entire sample ( $n = 81$ ) using particular medications<sup>a</sup>

Medication type	Pre-ECT number (%)	Post-ECT number (%)
Tricyclic or tetracyclic antidepressants	54 (66.7%)	58 (72.5%)
Selective serotonin re-uptake inhibitors (SSRIs) or monoamine oxidase inhibitors (MAOIs)	3 (3.7%)	2 (2.5%)
Antipsychotic medication	42 (51.9%)	27 (33.8%)
Sedative/hypnotic medication	52 (64.2%)	45 (56.3%)

<sup>a</sup> There were no significant differences in the use of any medication between pre- and post-treatment.

Table 2  
Side effects<sup>a</sup>

	Pts <70 years ( $n = 40$ )		Pt $\geq$ 70 years ( $n = 41$ )		All patients	
	Pre-ECT (%) ( $n = 34$ )	Post-ECT (%) ( $n = 36$ )	Pre-ECT (%) ( $n = 31$ )	Post-ECT (%) ( $n = 34$ )	Pre-ECT (%) ( $n = 65$ )	Post-ECT (%) ( $n = 70$ )
Muscle aches/pain	17.6	30.6	35.5	17.6	26.2	24.3
Constipation	38.2	36.1	67.7	50.0	52.3	42.9
Headache	26.5	27.8	16.1	29.4	21.5	28.6
Confusion	45.5*	27.8	41.9	54.5*	43.8*	40.6*
Clumsiness	33.3*	11.1	32.3	26.5	32.8*	18.6
Blurred vision	14.7	19.4	32.3	32.4	23.1	25.7
Dry mouth	64.7	38.9	61.3	52.9	63.1	45.7
Anterograde memory impaired	44.1	61.1	54.8	47.1	49.2	54.3
Retrograde memory impaired	29.4	22.2	25.8	23.5	27.7	22.9
Palpitations	23.5	13.9	29.0	8.8	26.2	11.4
Dizziness	28.1 <sup>#</sup>	27.8	45.2	20.6	36.5 <sup>#</sup>	24.3
Drowsiness	50.0	33.3	29.0	32.4	40.0	32.9
Nausea/vomiting	6.1*	13.9	12.9	5.9	9.4*	10.0
Neck stiffness	20.6	13.9	9.7	11.8	15.4	12.9
Urinary retention	14.7	19.4	19.4	8.8	16.9	14.3
Urinary incontinence	5.9	2.8	9.7	0	7.7	1.4
Faecal incontinence	0	0	9.7	0	4.6	0
Bruising	23.5	19.4	16.1	29.4	20.0	24.3
Neurological dysfunction	2.9	8.3	12.9	26.5	6.2	17.1
Medical complications	5.9	5.6	9.7	5.9	7.7	5.7
	Mean (S.D.)	Mean (S.D.)	Mean (S.D.)	Mean (S.D.)	Mean (S.D.)	Mean (S.D.)
Total burden score	9.9 (8.6)	8.3 (6.2)	12.0 (9.7)	9.4 (7.2)	10.9 (9.2)	8.8 (6.7)

<sup>a</sup> Missing data: for some side effects, the number of patients was one less indicated by \* or two less indicated by #.

in side effect burden after ECT (effect size 0.07; S.D. 0.85). There were no significant changes in the proportion of patients reporting a particular side effect.

Changes in the proportion reporting neurological impairment, dry mouth and palpitations approached significance. Neurological impairment increased by

9.5%, whilst dry mouth and palpitations decreased by 17.4 and 14.8%, respectively. Neurological signs and symptoms post-ECT comprised two cases each of tinnitus, tremor and parasthesia; and one each of ulnar nerve palsy, loss of voice, extra-pyramidal side effects, abnormal involuntary movements and clonic seizure.

We confirmed our second hypothesis about the link between side effects and depression in a number of ways. First, we found associations between side effect burden and HRSD whether measured pre-ECT ( $r = 0.38$ ,  $P = 0.005$ ) or post-ECT ( $r = 0.57$ ,  $P < 0.0001$ ). Secondly, there was a significant association between improvement in depression (pre–post effect size) and reduction in side effects (pre–post effect size) ( $r = 0.46$ ,  $P = 0.001$ ). The reduction in side effects was most pronounced in those with less depression post-ECT: post-treatment HRSD was negatively correlated with the pre–post effect size of side effect burden ( $r = -0.38$ ,  $P = 0.006$ ).

Most importantly, when change in depression was controlled for, the decline in side effect burden after ECT remained significant ( $F(1,51) = 7.79$ ,  $P = 0.007$ ). There was no significant correlation between duration of depression and side effect burden.

We examined the possible confounding effects of medication. There were no significant differences in side effect burden between those taking tricyclics and those not taking tricyclics either before or after ECT. However, those taking tricyclics or antipsychotics had a significantly higher side effect burden score prior to ECT (but not at post-ECT) than those taking neither ( $t(1,59) = -2.483$ ,  $P = 0.016$ ).

We confirmed our third hypothesis that there was no relationship between age and side effects. We repeated the above analyses after dichotomising the sample into patients aged below 70 years or 70 years and older. In neither group was there any significant change in side effect burden or in the proportion of patients reporting any particular side effect. Our results were also independent of patient gender.

### 3.4. Neuropsychological tests

We examined neuropsychological performance and again confirmed our first hypothesis. When scores were compared for subjects who had data on two occasions, there were no significant differences found between scores pre- and post-treatment or between pre-ECT and follow-up (Table 3). We repeated the analyses (i) only for patients who had data at all three assessments ( $n = 16–26$ ); and (ii) only for patients who did not develop dementia and who had data at all three assessments ( $n = 16–26$ ).

Both sets of analyses failed to show any significant differences over time.

Our expectation that cognitive impairment would be more pronounced directly after ECT in older patients was not confirmed. There were no significant differences when these analyses were repeated for all patients aged less than 70 years and for all those aged 70 years or greater or when male and female patients were examined separately. There were also no significant differences when the 12 patients who subsequently developed dementia were excluded, or when post and follow-up Hamilton depression scores were controlled for.

## 4. Discussion

All three hypotheses were confirmed, with qualifications. We replicated the finding that many of the so-called side effects of ECT appear to be largely symptoms of depression (Petinati and Rosenberg, 1984; Sackeim et al., 1993; Devanand et al., 1995). The prevalence of these symptoms was much the same after ECT as before ECT and correlated significantly with the level of depression. Moreover, improvement in depression correlated with reduction in these adverse effects. Similarly, performance on neuropsychological tasks did not change after ECT, or on longer follow-up. However we note that there were significant missing data on testing, reflecting the difficulty in performing neuropsychological assessment in profoundly depressed patients. Finally, the occurrence of ‘side effect’ symptoms was independent of age (and sex), the subsequent development of dementia or the persistence of depression. One cautionary note is the higher rate after ECT of neurological symptoms which may be clinically, if not statistically, significant. We were unable to pursue investigation of these symptoms further or to determine their persistence.

With the exception of the recent report from Tew et al. (1999), the size of our patient sample was similar to or larger than those previously reported (e.g., Rubin et al., 1993; Freeman et al., 1980; Taylor and Abrams, 1985; Abrams and Taylor, 1985). As regards the missing neuropsychological data, subjects who had not been tested were similar at index

Table 3  
Mean neuropsychological test scores (S.D., *n*) for all subjects with data<sup>a</sup>

	SDMT <sup>b</sup>	Trails A <sup>c</sup>	COWAT <sup>d</sup>	Simple RT <sup>e</sup>	Complex RT <sup>f</sup>
<i>Patients &lt;70 years</i>					
Pre-ECT mean	45.2	33.0	40.5	371.3	715.6
(S.D., <i>n</i> )	(12.4, 24)	(8.1, 31)	(14.1, 26)	(53.1, 29)	(74.7, 32)
Post-ECT mean	46.0	31.8	43.1	362.9	757.4
(S.D., <i>n</i> )	(10.8, 26)	(5.4, 30)	(13.2, 27)	(55.2, 31)	(199.5, 31)
Follow-up mean	44.9	32.4	42.4	351.1	698.2
(S.D., <i>n</i> )	(13.6, 21)	(8.3, 22)	(11.4, 20)	(56.8, 22)	(85.0, 21)
<i>Patients ≥70 years</i>					
Pre-ECT mean	29.9	70.9	35.3	479.1	900.9
(S.D., <i>n</i> )	(5.0, 16)	(21.4, 24)	(10.1, 20)	(128.2, 23)	(103.1, 21)
Post-ECT mean	28.8	75.2	33.5	525.2	911.8
(S.D., <i>n</i> )	(6.0, 19)	(24.3, 27)	(10.6, 26)	(138.3, 26)	(130.5, 26)
Follow-up mean	30.5	75.4	34.8	559.3	943.7
(S.D., <i>n</i> )	(7.0, 11)	(19.2, 19)	(13.7, 23)	(146.9, 22)	(116.4, 19)
<i>All patients</i>					
Pre-ECT mean	39.1	49.5	38.2	419.0	789.0
(S.D., <i>n</i> )	(12.6, 40)	(24.3, 55)	(12.7, 46)	(107.5, 52)	(125.7, 53)
Post-ECT mean	38.8	52.4	38.4	436.9	827.9
(S.D., <i>n</i> )	(12.5, 45)	(27.7, 57)	(12.8, 53)	(129.7, 57)	(186.9, 57)
Follow-up mean	40.0	52.4	38.3	455.2	814.8
(S.D., <i>n</i> )	(13.6, 32)	(26.0, 41)	(13.1, 43)	(152.3, 44)	(159.3, 40)

<sup>a</sup> Note: As comparisons were possible where patients had data on two occasions, numbers used in analyses differed from those presented in the table.

<sup>b</sup> Symbol Digit Modalities Test (SDMT).

<sup>c</sup> Trail making test part A (Trails A).

<sup>d</sup> Controlled Oral Word Association Test (COWAT) — 'FAS' version.

<sup>e</sup> Simple Reaction Time (Simple RT).

<sup>f</sup> Complex Reaction Time (Complex RT).

assessment clinically and demographically to those who had.

Methodological flaws in the study include the lack of a control group, the use of a memory scale that reflects new learning and acquisition rather than retention (Abrams, 1997), and the lack of detailed information on the ECT techniques which have been shown to influence reported ECT side effects (Sackeim et al., 1987; Devanand et al., 1994; Shapira et al., 1998). At the time of the study, Australian practice was to prescribe unilateral ECT preferentially unless there was a previous history of bilateral response or unless a course of unilateral ECT was proving unsuccessful. The lack of variance in ECT practice may have limited the potential of available ECT variables to account for side effect differences.

We considered the possibility that a real increase in side effect prevalence after ECT was masked by a

concurrent decline in depressive symptoms. We rejected this hypothesis because improvements in depression and in side effects were significantly correlated. Also, when we covaried for depression, we found a significant *decline* in side effect burden scores.

It is possible that medications confounded interpretation of the results. For instance, reduction in the use of tricyclic antidepressants and antipsychotics could have resulted in less blurred vision, urinary retention and drowsiness. Indeed, there was a relationship between use of *either* a tricyclic or antipsychotic and side effect burden pre-ECT. We do not put much weight on this for several reasons: (i) this latter relationship was not evident after ECT; (ii) there was no relationship between tricyclic use and side effect burden either pre- or post-ECT; and (iii) there were no significant differences in psychotropic

use from before to after ECT. (We note that this study was undertaken before selective serotonin uptake inhibitors had come into wide usage.)

## 5. Implications and conclusions

The effectiveness of ECT in treating depression in older patients is well substantiated (Cattan et al., 1990; Scott et al., 1992; Benbow, 1989; Tew et al., 1999; Brodaty et al., 2000). The present findings are reassuring. Despite popular misconceptions, ECT appears to be associated with few significant side effects. Older people do not appear to be more susceptible to side effects after ECT.

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